	Type	L #	Hits	Search Text	DBs	Time Stamp	Comments	Error Definition	Errors
1	BRS	L1	143	trp adj arg adj tyr	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/07/11 07:56			0
2	BRS	L2	0	ac adj trp adj arg adj tyr adj nh2	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/07/11 08:01			0
3	BRS	L3	0	acetyl same 1	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/07/11 08:01			0
4	BRS	L4	980	neuropeptide adj y	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/07/11 08:02			0
5	BRS	L5	399	(neuropeptide adj y) same (antagonist or agonist)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/07/11 08:02			0
6	BRS	L6	0	((neuropeptide adj y) same (antagonist or agonist)) same (trp adj arg adj tyr)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/07/11 08:02			0
7	BRS	L7	105472	(pharmaceutical or therapeutic) adj composition	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/07/11 08:03			0
8	BRS	L8	49882	carrier same ((pharmaceutical or therapeutic) adj composition)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/07/11 08:04			0
9	BRS	L9	1292	(carrier same ((pharmaceutical or therapeutic) adj composition)) same (magnesium adj carbonate)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/07/11 08:05			0

	Type	L #	Hits	Search Text	DBs	Time Stamp	Comments	Error Definition	Errors
10	BRS	L10	6190	(carrier same ((pharmaceutical or therapeutic) adj composition)) same (lactose)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/07/11 08:06			0
11	BRS	L11	1253	(carrier same ((pharmaceutical or therapeutic) adj composition)) same (magnesium adj carbonate) same lactose	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/07/11 08:06			0
12	BRS	L12	26	((carrier same ((pharmaceutical or therapeutic) adj composition)) same (magnesium adj carbonate) same lactose) same peptide	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/07/11 08:10			0
13	BRS	L13	46	cationized adj albumin	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/07/11 08:11			0
14	BRS	L14	4987	polylysine	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/07/11 08:12			0
15	BRS	L15	699	conjugate same ((cationized adj albumin) or polylysine)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/07/11 08:12			0
16	BRS	L16	24	biodegradable adj sustained-release	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/07/11 08:12			0

=> d his

(FILE 'HOME' ENTERED AT 08:17:04 ON 11 JUL 2002)

FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT
08:17:41 ON 11 JUL 2002

L1 5 S AC-TRP-ARG-TYR-NH2
L2 1 DUPLICATE REMOVE L1 (4 DUPLICATES REMOVED)
L3 43962 S NEUROPEPTIDE Y
L4 3464 S L3 (P) (AGONIST OR ANTIGONIST)
L5 6 S L4 (P) TRIPEPTIDE
L6 2 DUPLICATE REMOVE L5 (4 DUPLICATES REMOVED)
L7 2 S L6 NOT L2
L8 20 S L4 (P) (ALBUMIN OR POLYLYSINE)
L9 5 DUPLICATE REMOVE L8 (15 DUPLICATES REMOVED)

=> log y

FILE 'HOME' ENTERED AT 08:17:04 ON 11 JUL 2002

=> file medline caplus biosis enbase scisearch agricola
'ENBASE' IS NOT A VALID FILE NAME
Enter "HELP FILE NAMES" at an arrow prompt (=>) for a list of files
that are available. If you have requested multiple files, you can
specify a corrected file name or you can enter "IGNORE" to continue
accessing the remaining file names entered.
ENTER A FILE NAME OR (IGNORE):embase
COST IN U.S. DOLLARS

	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'MEDLINE' ENTERED AT 08:17:41 ON 11 JUL 2002

FILE 'CAPLUS' ENTERED AT 08:17:41 ON 11 JUL 2002
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FILE 'AGRICOLA' ENTERED AT 08:17:41 ON 11 JUL 2002

=> s ac-trp-arg-tyr-nh2
L1 5 AC-TRP-ARG-TYR-NH2

=> duplicate l1
ENTER REMOVE, IDENTIFY, ONLY, OR (?):remove
DUPLICATE PREFERENCE IS 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH'
KEEP DUPLICATES FROM MORE THAN ONE FILE? Y/(N):n
PROCESSING COMPLETED FOR L1
L2 1 DUPLICATE REMOVE L1 (4 DUPLICATES REMOVED)

=> d l2 1 ibib abs

L2 ANSWER 1 OF 1 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 1998398379 MEDLINE
DOCUMENT NUMBER: 98398379 PubMed ID: 9729264
TITLE: WRYamide, a NPY-based tripeptide that antagonizes feeding in rats.
AUTHOR: Chance W T; Tao Z; Sheriff S; Balasubramaniam A
CORPORATE SOURCE: Department of Surgery, University of Cincinnati Medical Center, 231 Bethesda Avenue, Cincinnati, OH 45267, USA.
CONTRACT NUMBER: GM 47122 (NIGMS)
SOURCE: BRAIN RESEARCH, (1998 Aug 24) 803 (1-2) 39-43.
Journal code: 0045503. ISSN: 0006-8993.
PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199905
ENTRY DATE: Entered STN: 19990607
Last Updated on STN: 19990607
Entered Medline: 19990526

AB Modifications of (D-Trp32) neuropeptide Y (NPY) led to the development of potential peptide-based lower molecular weight (500-800 Da) NPY feeding antagonists. One compound, WRYamide (N- ***Ac*** - ***Trp*** - ***Arg*** - ***Tyr*** - ***NH2***), blocked NPY-induced feeding for 1 to 4 h when injected intrahypothalamically (i.h.t.) at 1 to 40 microgram. Schedule-induced feeding was also antagonized for up to 24 h by 20 microgram of WRYamide, i.h.t. Injection of 2.5 mg/kg (1 mg/rat) of

WRYamide, i.v., also reduced significantly schedule-induced feeding for 4 h. A conditioned taste aversion could not be classically conditioned to saccharin using WRYamide as the unconditioned stimulus. These results may lead to the development of systemically active anti-obesity drugs. Copyright 1998 Elsevier Science B.V.

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=> s neuropeptide y
L3        43962 NEUROPEPTIDE Y

=> s l3 (agonist or antigonist)
MISSING OPERATOR 'L15 (AGONIST'
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> s l3 (p) (agonist or antigonist)
L4         3464 L3 (P) (AGONIST OR ANTIGONIST)

=> s l4 (p) tripeptide
L5            6 L4 (P) TRIPEPTIDE

=> duplicate remove l5
DUPLICATE PREFERENCE IS 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L5
L6             2 DUPLICATE REMOVE L5 (4 DUPLICATES REMOVED)

=> s l6 not l2
L7             2 L6 NOT L2

=> d l7 1-2 ibib abs

L7   ANSWER 1 OF 2     MEDLINE
ACCESSION NUMBER:    1999089557     MEDLINE
DOCUMENT NUMBER:     99089557   PubMed ID: 9874161
TITLE:               BIBP 3226 inhibition of nicotinic receptor mediated
                     chromaffin cell secretion.
AUTHOR:              Zhang P; Zheng J; Hexum T D
CORPORATE SOURCE:    Department of Pharmacology, University of Nebraska Medical
                     Center, Omaha 68198-6260, USA.
SOURCE:              EUROPEAN JOURNAL OF PHARMACOLOGY, (1998 Dec 4) 362 (2-3)
                     121-5.
                     Journal code: 1254354. ISSN: 0014-2999.
PUB. COUNTRY:        Netherlands
                     Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:            English
FILE SEGMENT:        Priority Journals
ENTRY MONTH:         199903
ENTRY DATE:          Entered STN: 19990326
                     Last Updated on STN: 19990326
                     Entered Medline: 19990318
AB   (R)-N 2-(diphenacetyl)-N-[(4-hydroxyphenyl)methyl]-argininamide (BIBP
     3226) is a selective   ***neuropeptide***     ***Y***   Y1 receptor
     antagonist with structural similarity to the C-terminal   ***tripeptide***
     of   ***neuropeptide***     ***Y***  . Based on this similarity we
     questioned whether BIBP 3226 could act as an   ***agonist***  . Incubation
     of BIBP 3226 with bovine chromaffin cells in culture results in the
     inhibition of nicotinic receptor-stimulated catecholamine secretion (IC50
     = 2.4 microM). The effect of BIBP 3226 is independent of
       ***neuropeptide***     ***Y***   action since the presence of
       ***neuropeptide***     ***Y***   in the culture medium does not alter the
     effect of BIBP 3226. BIBP 3226 decreased the efficacy of the nicotinic
     receptor   ***agonist***  , 1,1-dimethyl-4-phenylpiperizinium (DMPP), but
     did not change its potency suggesting non-competitive inhibition. BIBP
     3226 has a similar effect on nicotinic receptor-stimulated 45Ca2+ influx.
     BIBP 3226 does not inhibit [3H]norepinephrine release induced by high K+
     and its effect is not pertussis toxin-sensitive. We conclude that not only
     can BIBP 3226 act as a   ***neuropeptide***     ***Y***   receptor
     antagonist in bovine chromaffin cells but also act as an   ***agonist***
     and inhibit catecholamine secretion.

L7   ANSWER 2 OF 2  CAPLUS  COPYRIGHT 2002 ACS
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ACCESSION NUMBER: 2000:31[redacted]42 CAPLUS
DOCUMENT NUMBER: 132:88[redacted]
TITLE: Neuropeptide Y agonist and antagonist peptides for control of appetite, blood pressure, cardiovascular response, libido, and circadian rhythm
INVENTOR(S): Balasubramanium, Ambikaipakan; Chance, William T.
PATENT ASSIGNEE(S): University of Cincinnati, USA
SOURCE: U.S., 17 pp.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6013633	A	20000111	US 1997-907403	19970807
US 6235718	B1	20010522	US 1999-449914	19991202

PRIORITY APPLN. INFO.: US 1997-907403 A3 19970807
OTHER SOURCE(S): MARPAT 132:88195
AB Dipeptides and ***tripeptides*** , and methods for pharmaceutical treatment of mammals using analogs of such dipeptides and ***tripeptides*** , are provided. More specifically, the invention relates to ***tripeptides*** and their analogs, to pharmaceutical compns. contg. such dipeptides and ***tripeptides*** , and to methods of treatment of mammals using such dipeptides and ***tripeptides*** . In addn., the invention relates to methods of treatment of mammals using such dipeptides and ***tripeptides*** for control of appetite, blood pressure, cardiovascular response, libido, and circadian rhythm. The compds. of the invention are ***neuropeptide*** ***Y*** receptor ***agonists*** and antagonists.
REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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=> d his

     (FILE 'HOME' ENTERED AT 08:17:04 ON 11 JUL 2002)

     FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT
     08:17:41 ON 11 JUL 2002
L1             5 S AC-TRP-ARG-TYR-NH2
L2             1 DUPLICATE REMOVE L1 (4 DUPLICATES REMOVED)
L3         43962 S NEUROPEPTIDE Y
L4          3464 S L3 (P) (AGONIST OR ANTIGONIST)
L5             6 S L4 (P) TRIPEPTIDE
L6             2 DUPLICATE REMOVE L5 (4 DUPLICATES REMOVED)
L7             2 S L6 NOT L2

=> s l4 (p) (albumin or polylysine)
L8            20 L4 (P) (ALBUMIN OR POLYLYSINE)

=> duplicate remove l8
DUPLICATE PREFERENCE IS 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L8
L9             5 DUPLICATE REMOVE L8 (15 DUPLICATES REMOVED)

=> d l9 1-5 ibib abs

L9   ANSWER 1 OF 5  SCISEARCH  COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER:    2001:212184  SCISEARCH
THE GENUINE ARTICLE: 404WQ
TITLE:               Designing of an orally active complement C3a agonist
                     peptide with anti-analgesic and anti-amnesic activity
AUTHOR:              Jinsmaa Y; Takenaka Y; Yoshikawa M (Reprint)
CORPORATE SOURCE:    Kyoto Univ, Food Sci Res Inst, Uji, Kyoto 6110011, Japan
                     (Reprint)
COUNTRY OF AUTHOR:   Japan
SOURCE:              PEPTIDES, (JAN 2001) Vol. 22, No. 1, pp. 25-32.
                     Publisher: ELSEVIER SCIENCE INC, 655 AVENUE OF THE
                     AMERICAS, NEW YORK, NY 10010 USA.
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ISSN: 0196-9781.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 40
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Complement C3a is an anti-opioid peptide, having anti-analgesic and anti-amnesic effects after intracerebroventricular administration. However, the peptide is inactive after oral administration. Orally active C3a agonist peptide was designed based on the structure of oryzatensin, a C3a agonist peptide derived from rice albumin. Tyr-Pro-Leu-Pro-Agr. a pentapeptide at the carboxyl terminus of oryzatensin is the minimally essential structure for exerting C3a activity. Due to the affinity fur mu -opioid receptor, both oryzatensin and Tyr-Pro-Leu-Pro-Arg showed analgesia after intracerebroventricular administration in mice which was blocked by the opioid antagonist naloxone. Tyr-Pro-Leu-Pro-Arg lust opioid activity by substitution the amino terminus tyrosine with other hydrophobic residues. Among the newly designed peptides, Trp-Pro-Leu-Pro-Arg was found to possess the strongest C3a activity. The peptide antagonized morphine-induced analgesia at 300 mg/kg after oral administration and also improved scopolamine- and ischemia-induced amnesia in a step-through passive avoidance test. (C) 2001 Elsevier Science inc. All rights reserved.

L9 ANSWER 2 OF 5 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 1998118999 MEDLINE
DOCUMENT NUMBER: 98118999 PubMed ID: 9457655
TITLE: Sympathetic and parasympathetic interaction in vascular and secretory control of the nasal mucosa in anaesthetized dogs.
AUTHOR: Revington M; Lacroix J S; Potter E K
CORPORATE SOURCE: Prince of Wales Medical Research Institute, Prince of Wales Hospital, Sydney, NSW, Australia.
SOURCE: JOURNAL OF PHYSIOLOGY, (1997 Dec 15) 505 (Pt 3) 823-31.
Journal code: 0266262. ISSN: 0022-3751.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199803
ENTRY DATE: Entered STN: 19980319
Last Updated on STN: 19980319
Entered Medline: 19980312

AB 1. In dogs anaesthetized with pentobarbitone, electrical stimulation of the parasympathetic nerve fibres to the nasal mucosa evoked frequency dependent increases in both nasal arterial blood flow and nasal secretion. Blood flow was measured using a transonic flow probe placed around the artery. 2. Sympathetic nerve stimulation for 3 min at 10 Hz evoked significant and prolonged (> 30 min) attenuation of the vasodilator and secretory responses to subsequent parasympathetic stimulation. 3. Intravenous and intranasal administration of the ***neuropeptide*** ***Y*** (NPY) analogue N-acetyl [Leu28,Leu31] NPY 24-36, a selective NPY Y2 receptor ***agonist*** (20 nmol kg-1), significantly attenuated both vasodilator and secretory effects of subsequent parasympathetic nerve stimulation. When given intravenously, the inhibitory effect of this Y2 receptor ***agonist*** on vascular and secretory effects of parasympathetic nerve stimulation was rapid in onset (5 min) and lasted for more than 60 min. The modulatory effect of the Y2 receptor ***agonist*** was also seen with intranasal administration, but was slower in onset (15 min), and lasted less than 45 min. The effects of the intranasal pretreatment with the Y2 receptor ***agonist*** were significantly prolonged in the presence of the endopeptidase inhibitor phosphoramidon (10 nM). 4. Atropine pretreatment did not significantly reduce the change in vascular conductance evoked by parasympathetic nerve stimulation. Subsequent pretreatment with the NPY Y2 receptor ***agonist*** N-acetyl [Leu28,Leu31] NPY 24-36 reduced the stimulation induced increase in conductance by 30%. Nasal secretion was reduced by 70% following pretreatment with atropine and a further 30% by pretreatment with the NPY Y2 receptor ***agonist*** . Dose dependent vasodilator and secretory effects of local intra-arterial infusion of acetylcholine and vasoactive intestinal peptide were not modified by the NPY Y2 ***agonist*** . 5. Total protein and ***albumin*** concentration were measured in nasal lavage fluid collected after nerve stimulation. Atropine

pretreatment increased the percentage of the total protein that was ***albumin*** in nasal lavage fluid. Neither sympathetic nerve stimulation nor Y2 receptor ***agonist*** pretreatment further modified the ***albumin*** exudation (a marker of vascular permeability) in nasal fluid lavage collected after parasympathetic nerve stimulation. 6. We propose that sympathetic nerve stimulation releases NPY, which acts on Y2 receptors, probably located on parasympathetic nerve endings, to attenuate both vasodilatation and nasal secretion evoked by subsequent parasympathetic nerve stimulation. This effect is also observed after pretreatment with the Y2-selective NPY analogue N-acetyl [Leu28,Leu31] NPY 24-36.

L9 ANSWER 3 OF 5 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 97041131 MEDLINE
DOCUMENT NUMBER: 97041131 PubMed ID: 8886402
TITLE: Neuropeptide Y Y2 receptor-mediated attenuation of neurogenic plasma extravasation acting through pertussis toxin-sensitive mechanisms.
AUTHOR: Yu X J; Moskowitz M A
CORPORATE SOURCE: Massachusetts General Hospital, Harvard Medical School, Charlestown 02129, USA.
SOURCE: BRITISH JOURNAL OF PHARMACOLOGY, (1996 Sep) 119 (2) 229-32. Journal code: 7502536. ISSN: 0007-1188.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199702
ENTRY DATE: Entered STN: 19970219
Last Updated on STN: 19970219
Entered Medline: 19970206
AB 1. The effects of ***neuropeptide*** ***Y*** (NPY) receptor ***agonists*** (administered intravenously) were examined on plasma protein ([125I]-bovine serum ***albumin***) leakage within dura mater evoked by unilateral trigeminal ganglion stimulation (0.6 mA, 5 ms, 5 Hz, 5 min), capsaicin (1 mumol kg-1, i.v.) or substance P (1 nmol kg-1, i.v.) in anaesthetized Sprague-Dawley rats. 2. NPY (EC50: 5.6 nmol kg-1) and NPY fragment 13-36 [NPY (13-36)] (ED50: 4.3 nmol kg-1), an NPY Y2 receptor ***agonist*** , dose-dependently attenuated [125I]-bovine serum ***albumin*** extravasation from meningeal vessels when administered 10 min prior to electrical stimulation. [Leu31, Pro34]-NPY, an NPY Y1 and Y3 receptor ***agonist*** , inhibited the response at a higher dose only (23 nmol kg-1) ($P < 0.05$). 3. NPY also significantly decreased plasma protein extravasation induced by capsaicin (1 mumol kg-1) but not by substance P (1 nmol kg-1). 4. Pertussis toxin (20 micrograms kg-1, administered intracisternally 48 h prior to stimulation) blocked completely the inhibitory effect of NPY and NPY (13-36) but did not inhibit extravasation alone. 5. We conclude that NPY inhibits neurogenically-mediated plasma protein extravasation acting through presynaptic pertussis toxin-sensitive NPY Y2 receptors, possibly by inhibition of neuropeptide release from perivascular trigeminovascular afferents.

L9 ANSWER 4 OF 5 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 94247727 MEDLINE
DOCUMENT NUMBER: 94247727 PubMed ID: 8190350
TITLE: Neuropeptide Y (NPY)- and vasoactive intestinal peptide (VIP)-induced aldosterone secretion by rat capsule/glomerular zone could be mediated by catecholamines via beta 1 adrenergic receptors.
AUTHOR: Bernet F; Bernard J; Laborie C; Montel V; Maubert E; Dupouy J P
CORPORATE SOURCE: Neuroendocrinologie du Developpement, Universite de Lille, Villeneuve d'Ascq, France.
SOURCE: NEUROSCIENCE LETTERS, (1994 Jan 17) 166 (1) 109-12. Journal code: 7600130. ISSN: 0304-3940.
PUB. COUNTRY: Ireland
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199406
ENTRY DATE: Entered STN: 19940629

Last Updated on STN: 19940629
Entered Medline: 19940620

AB The effects of two ***Neuropeptide*** ***Y*** (NPY) analogs (Y1- and Y2-type) and vasoactive intestinal peptide (VIP) on both catecholamine (adrenaline and noradrenaline) release and aldosterone production by rat adrenal capsule/glomerular zone, have been investigated in vitro. The adrenal capsule/glomerular zones, collected from adult male rats, were incubated in a medium (Krebs-Ringer bicarbonate buffer supplemented with glucose and bovine serum ***albumin***) containing or not one of the following synthetic peptides: human Leu31,Pro34-NPY (an ***agonist*** of the Y1-type receptors), human/porcine NPY18-36 (an ***agonist*** of the Y2-type receptors) and VIP at the concentration of 10(-7) M, associated or not with 10(-7) M atenolol (a beta 1 adrenergic antagonist) or ICI-118,551 hydrochloride (a beta 2 adrenergic antagonist). The two NPY analogs as well as the VIP stimulated the release of catecholamines and of aldosterone. The beta 1 adrenergic antagonist, but not the beta 2 one, which failed to affect basal aldosterone production when given alone, prevented NPY18-36-, Leu31,Pro34-NPY- or VIP-induced aldosterone secretion. Present data support the hypothesis that adrenaline and/or noradrenaline could mediate the effects of both NPY and VIP on aldosterone secretion via beta 1 adrenergic receptors; alternatively, the steroidogenic effect of NPY or VIP could be related to direct interaction between NPY- or VIP-specific binding sites, present on the capsule/glomerular zone of the rat adrenal cortex, and beta 1 adrenergic receptors. Then the NPYergic, VIPergic and catecholaminergic innervation of the adrenal cortex, previously characterized by immunohistochemistry, may be a potent stimulatory element in the nervous control of the aldosterone secretion.

L9 ANSWER 5 OF 5 MEDLINE DUPLICATE 4

ACCESSION NUMBER: 93115141 MEDLINE
DOCUMENT NUMBER: 93115141 PubMed ID: 1282125
TITLE: Neuropeptide Y is a vasoconstrictor in human nasal mucosa.
AUTHOR: Baraniuk J N; Silver P B; Kaliner M A; Barnes P J
CORPORATE SOURCE: Department of Medicine, Georgetown University, Washington, DC 20007.
SOURCE: JOURNAL OF APPLIED PHYSIOLOGY, (1992 Nov) 73 (5) 1867-72. Journal code: 8502536. ISSN: 8750-7587.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199302
ENTRY DATE: Entered STN: 19930219
Last Updated on STN: 19960129
Entered Medline: 19930201

AB ***Neuropeptide*** ***Y*** (NPY) is a neurotransmitter in sympathetic nerve fibers in human nasal mucosa. Like norepinephrine, NPY acts as a vasoconstrictor. An established method of nasal provocation was used to determine the effects of topically applied NPY on nasal resistance to airflow measured by anterior rhinomanometry, the protein content of nasal secretions, and the protein content of bradykinin-induced secretions. NPY (2.3 nmol) reduced the resistance to inspiratory airflow by 57 +/- 18% ($P < 0.001$) in 10 normal subjects and by 50 +/- 17% ($P < 0.05$) in 12 subjects with perennial rhinitis. In nasal provocations, NPY in doses of 0.1-10 nmol had no effect on vascular (***albumin***), glandular (lysozyme, glycoconjugate), or total proteins present in lavaged nasal secretions. Because the vasoconstrictor properties of NPY may only be apparent in the presence of increased vascular permeability and ***albumin*** exudation, bradykinin (BK) nasal provocation was performed. BK (500 nmol) significantly increase total protein (10- to 20-fold), ***albumin*** (10- to 30-fold), and glycoconjugate (2- to 5-fold) in lavage fluid. NPY (2.3 nmol) reduced BK-induced total protein by 59 +/- 15% ($P < 0.05$) and ***albumin*** by 63 +/- 17% ($P < 0.02$) but had no significant effect on glandular secretion. Therefore exogenous administration of NPY to the human nasal mucosa reduced nasal airflow resistance and ***albumin*** exudation without affecting submucosal gland secretion. NPY ***agonists*** may be useful for the treatment of mucosal diseases characterized by vasodilation, vascular permeability, and plasma exudation.

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=> d his

     (FILE 'HOME' ENTERED AT 08:17:04 ON 11 JUL 2002)

     FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT
     08:17:41 ON 11 JUL 2002
L1             5 S AC-TRP-ARG-TYR-NH2
L2             1 DUPLICATE REMOVE L1 (4 DUPLICATES REMOVED)
L3         43962 S NEUROPEPTIDE Y
L4          3464 S L3 (P) (AGONIST OR ANTIGONIST)
L5             6 S L4 (P) TRIPEPTIDE
L6             2 DUPLICATE REMOVE L5 (4 DUPLICATES REMOVED)
L7             2 S L6 NOT L2
L8            20 S L4 (P) (ALBUMIN OR POLYLYSINE)
L9             5 DUPLICATE REMOVE L8 (15 DUPLICATES REMOVED)

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COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
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FULL ESTIMATED COST                                   36.42      36.63

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
                                                      ENTRY    SESSION
CA SUBSCRIBER PRICE                                   -0.62      -0.62

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